

M. D. T H E S I S.

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Subject:-

A supposed case of Madura Foot, occurring in a
lad of Scottish parentage, who was never furth of
Scotland.

March 28th, 1913.



I N D E X.

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Appended are (1) side view of foot and leg,
and (2) front view of foot and leg.

Up till December, 1910, I had regarded the subject of Madura Foot with only a general interest as a disease little likely to be met with in a practice such as mine. I looked on it as a malady believed to occur only in tropical climates, and rarely even there; whose pathology is little understood and for which no curative treatment is known. From that day till this the subject has been much on my mind. I have had under treatment a lad, at that time 13 years of age, who had never been out of this country, and who was not known to have been exposed in any way to tropical infection. Yet he developed a train of symptoms unlike anything I had ever seen before; the suggestion was made that the disease might be Madura Foot; but every practitioner familiar with Madura Foot who saw the case pronounced it unlike any such case he had ever seen; apart from this suggestion no practitioner of experience would venture the slightest surmise as to the nature of the disease, confessing that he had never seen anything in the least resembling it. As I had never before met with a case in which not the slightest suggestion of a diagnosis could be offered, the case has appealed to me with an interest beyond that possessed by any other case I have hitherto met with.

I propose (1) to recount the history of the case referred to; (2) to give a brief resume of the literature of Madura Foot; (3) and then to offer some considerations for and against the diagnosis of Madura Foot in this case.

H i s t o r y.

On 14th December, 1910, I was asked to examine a lad, named James Law Stirling, aged 13, who was said to be walking lame. I had to confess that I could see no evidence of lameness, as examined walking on a carpet. It was explained that the slight lameness was more noticeable when the lad walked on a rough road. A fortnight later I was again requested to see him, as the lameness was becoming more marked. I could now detect, even on the carpet, that Stirling put down the right foot with more care than the left. With the exception of a slight glossiness on the outer aspect of the right foot over the proximal end of the metatarsal bone (fifth) I could detect no sign of lesion.

Ten days later, on the 7th January, 1911, I examined the foot again. I now found that the glossiness on the outer aspect of the foot, above referred to, had developed into a raised, reddened, glossy surface, about two-thirds of an inch in diameter. This raised surface was tender to the touch. But, in addition, there was now decided pain all

along the under surface of the first metatarsal bone; the bone being swollen and tender to such a degree as to remind one of the earlier periods of periostitis of such a bone.

During the next two or three weeks an attempt was made to deal with these symptoms on ordinary lines. Rest in bed was enjoined, poultices were applied, phenacetine and aspirine were given for the relief of the pain, and, afterwards, ointment of biniodide of mercury was ordered to be rubbed into the skin as a deobstruent.

By this time great atrophy of the muscles of the leg had appeared. It was suggested that this was due to disuse; but it seemed to me to be both more complete and more rapid in development than could be explained on that hypothesis.

The result of the above treatment could not be said to be satisfactory. A fortnight after it was begun the state of matters was, that the glistening surface on the outer aspect of the foot now appeared as a tuberculated surface, with a larger and a smaller tubercle, exquisitely painful to touch; the first metatarsal bone was fully as inflamed as at the beginning of treatment; a small tubercle was appearing on the inner edge of the nail of the first toe, just at the junction of the nail with the skin,

and the first toe was showing a very marked tendency to remain in a condition of extreme extension. The whole foot, indeed, seemed to be in a condition of hyperaesthesia, for it had now become necessary to protect the whole foot from the pressure of the bed clothes by placing it at night inside a bandbox.

As I was dissatisfied with the existing ~~at~~ state of matters, I suggested that the opinion of Sir Hector Cameron should be obtained. As some reluctance was shown to the taking of this step, I pointed out to the parents, (1) that I was convinced that this was a case such as I had never seen before, (2) that the symptoms were, in my opinion, a unity, and that the condition of the outer aspect of the foot and the condition of the great toe were not a mere coincidence, (3) that I believed the condition to be due to some blood condition and not to any accidental violence; the suggestion having been offered that the condition was due to a ~~kick~~ that Stirling had sustained on the outer aspect of the foot while playing at football about a fortnight before I first saw him.

Sir Hector Cameron was of opinion ^{that} ~~was~~ the case was one of Hallux Rigidus. Authorities seem divided as to the ~~case~~ exact meaning of this term.

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In any case, Hallux Rigidus is evidently the name of a symptom and not of a pathological entity. This consultation took place on 18th March, 1911. Certain treatment was recommended of a soothing nature, and this was persisted in till April 24th. As no improvement was apparent, Sir Hector Cameron was again consulted. He at once saw that Hallux Rigidus was no explanation of the case, and advised that the tubercles on the outer aspect of the foot should be curetted.

At this stage there was a red, glistening area, about the size of a crown piece, on the outer aspect of the foot, with two elevated tubercles, one much larger than the other, in the centre of the inflamed area. The inflammation of the foot over the first metatarsal bone was very great; there was much inflammation on the upper surface of the great toe, at the junction of the skin and nail, and the little toe was much inflamed and swollen, with a boggy feeling.

The curetting was carried out under Chloroform on May 2nd. A small amount of matter was found inside the tubercles. Unfortunately the curetted matter was brought in contact with strong Carbolic Lotion before being submitted to microscopical examination and the results of examination were negative.

In the months of July and August Stirling was taken to Arran for change of air. Just before Stirling left, Sir Hector Cameron again saw the case and admitted that there was no sign of improvement. About a fortnight after the last mentioned interview, Sir Hector drew my attention to a case recently published by Dr. Norman Walker, of a rare form of bacterial disease of the skin, and suggested that Dr. Walker might be shown the case. The lad was taken to Edinburgh on July 15th, and was seen by Dr. Walker, in conjunction with Dr. Ritchie, of the Research Laboratory of the R. C. P. E. They were unable to hazard even a suggestion as to diagnosis, but were of opinion that the case was not one of Madura Foot. By this time the inflammation at the junction of the skin and nail of the great toe had broken down into an ulcer. Dr. Ritchie took a swab from this for examination, as well as material for the preparation, if possible, of a vaccine. While unable to offer a positive diagnosis, these gentlemen advised that the case should be treated with Iodide of Potassium, in thirty grain doses, three times a day, and that trial should be made of a self-vaccine. Ten flasks of this

vaccine were supplied. The contents of these flasks were administered at intervals of three weeks, one eighth of the first flask being administered, one fourth of the second, one half of the third, and thereafter the full contents of the flask. As the morbid process was being subjected both to the action of Iodide of Potassium and of the vaccine, it was difficult to determine which of the two was the one that produced benefit. It was the case, however, that during the months that this combined treatment was being carried on, the outward manifestations of the disease became stationary and even seemed to improve. The administration of the vaccines was not accompanied by, or followed by any reaction. The administration of such large doses of Iodide of Potassium made more striking, what had all along been a very striking feature of the case, the excellent health enjoyed by the lad. There was even an almost entire absence of Acne. Dr. Norman Walker also suggested the taking of a photograph of the foot. This was done on July 26th, and the accompanying photographs (1) and (2) are the representations of the foot at that date. They show the ulcerated surfaces on the outer aspect of the

healed daily, but he could not bear any pressure on the sole, such as would have been necessary in walking.

foot and at the inner aspect of the great toe. Unfortunately, the distinctness is obscured by the staining of the skin by the Iodine, a preparation of which had been used for a few days before the photographs were taken. It will be observed from the photographs that the expansion of the great toe, referred to early in the case, was still a marked feature of the case. The extreme atrophy of the muscles of the leg, mentioned as an early feature of the case, is here very prominently seen. But from about six weeks from the first complaint of tenderness in the foot, the atrophy had not increased.

The use of the vaccines extended over the period from October 14th, 1911, to March 29th, 1912. After that date the use of the vaccine was discontinued, but the administration of the Iodide of Potassium in half-dram doses was continued. The morbid process during that time certainly appeared to be at least stationary.

As the summer had again come round, it was arranged that the lad should be taken to Arran for two months. During his stay there his general health became even robust, and he was able to row for several hours daily, but he could not bear any pressure on the sole, such as would have been necessary in walking.

Whether as the result of the greater liberty taken in Arran, or due to the cessation of the vaccines, at this period the disease began to manifest itself at various points, The second, third and fourth toes becoming affected. The manifestation consisted in the development of small nodular masses, and, curiously enough, in each case, the tubercle was situated at the junction of the skin and nail. Whether it was due to the system being saturated with Iodide of Potassium and vaccines, but in each instance these tubercles were stunted in growth, and, with the exception of the second toe, gradually aborted and shrivelled without discharging any matter. These tubercles were not at any time so tender to the touch as the earlier manifestations of the disease had been.

Stirling spent July, 1912, at Benderloch. Whether the local conditions did not suit him, or whether too much exercise was taken- certain it is that the foot looked decidedly worse at his return at the end of July.

The state of matters now was that the atrophy of the leg was still as great as ever. The ulceration at the side of the nail of the great toe had all but healed, but there was no tendency for healthy nail to grow on the side of the toe where the ulcer had been. The discharges from the point of the second toe; and, as will

tenderness of the under surface of the first metatarsal bone had altogether disappeared, the unhealthy surface on the outer aspect of the foot where the disease had first appeared, was now all but well. The tubercle, however, at the point of the second toe had now broken down and the whole end of the toe was occupied by a purple, fungating, bleeding mass, which appeared to be pretty tender.

Coincidentally with this development of the disease in the second toe, the little toe became so swollen, bloated, purple and tense, as to make the third and fourth toes seem quite small by comparison. The little toe became at the same time a good deal everted, and all power of approximation to the other toes seemed lost.

It seems to me that the course of the disease up to this point might be divided into three stages. First, the stage of Invasion, marked by the outbreaks on the outer aspect of the foot and on the great toe, accompanied by swelling and tenderness of the first metatarsal bone; Second, a stage of Extension, during which the edges of the nails of the second, third, and fourth toes were attacked, but the disease seemed at this stage of diminished virulence; Third, a stage of Aggravation, during which the deeper structures became involved, leading to the swelling of the little toe, persistent fungous discharges from the point of the second toe; and, as will

be immediately seen, absorption of mineral matter from the metatarsal and phalangeal bones of the little toe, as well as of the proximal end of the metatarsal bone of the fourth toe.

About this time a young veterinary surgeon asked leave to prepare a slide from the discharge from the point of the second toe. Having done so he drew my attention to the remarkable resemblance between the slide and the figure of *Streptothrix Madurae* as given in Muir and Nitchie's Bacteriology. The resemblance seemed to me so striking that I wrote to Dr. Ritchie, of Edinburgh, asking his opinion on the subject. Dr. Ritchie replied that the bacillus with which he had dealt was not a *Streptothrix*.

In July, 1912, Stirling was seen in Edinburgh once again by Dr. Norman Walker and Dr. Ritchie. Dr. Ritchie abstracted some of the fluid from the most bloated part of the fifth toe with the view of obtaining a culture, but the results were negative. At this interview these gentlemen expressed themselves as unable to form an opinion as to the nature of the disease, but later Dr. Walker stated that further consideration had led Dr. Ritchie and himself to be much impressed with the resemblance the foot was beginning to manifest to the descriptions of Madura Foot. They had no new suggestion to offer as to the treatment; they hoped to resume the use of

a self-vaccine; but, as has been stated, the results of their attempted culture were negative.

During July, 1912, Dr. Macphail and Mrs. Dr. Macphail, both of whom had lengthened Indian experience and who had seen cases of Madura Foot, examined the foot. They were both of opinion that, if the case were one of Madura Foot, it was unlike any they had seen. Colonel Shearer, C.B., D.S.O., I.M.S., who had seen one or more cases of the disease, also examined the case. He also admitted himself unable to offer a diagnosis; but concurred in the opinion that it bore no resemblance to Madura Foot as he had seen it.

It was not surprising that, after experience of this multitude of medical counsellors, unable to formulate a diagnosis, the relatives should have listened to extra-professional advisers, of many shades of opinion. The result was an amusing interlude which should be mentioned as essential to the completeness of the history of the case.

Some relatives of the patient had become imbued with the possibilities of Osteopathy, and were anxious to place the lad in the care of a registered practitioner professing that system. With my consent he saw the lad. He refused so much as to examine the foot; but, from an examination of the spine, he declared that the case was exactly as he had expected, and such as he had successfully treated before by spinal manipulation. Although

invited to examine the foot, he declined; and although pressed to examine it, he refused. When I was informed of his undertaking to cure the case, (the time occupied was to be three months, three sittings a week, at one guinea a sitting, payable in advance,) I requested an interview with the Osteopathist. I had at an early stage of the disease suggested the idea of some interference with trophic influence of nerves, and I expressed myself as ready to be convinced. The interview was granted with very ill grace. The pathology of the case was declared to be that, in consequence of a slight fall three years before, (of which the boy had hitherto said nothing) absorption of the intervertebral discs had occurred, and that the cure was to be effected by "spinal manipulation". Questioned as to the nerve which he believed to be at fault, he hesitated for a minute or two and then named the Sciatic. Asked by Colonel Shearer about the point at which the Sciatic had been the subject of lesion, he pointed to a spot about ten or twelve inches too high. He compared the muscles on the two sides of the spine in the dorsal region; and, on the patient yielding to touch, adduced this as a proof of the tenderness of the spinal muscle. I failed on trial to elicit this tenderness; and, on afterwards asking the patient the cause of my failure, he informed me that the Osteopath had tickled him.

As certain relatives were enamoured of the idea, it was necessary to convince them of the futility of the proposed treatment, and, for that purpose, the accompanying skiagrams were taken. The spinal skiagram shows the spine to be normal. The dark body shown at the side had no connection with the ~~side~~^{spine}, for it was observed to move during respiration. The skiagram of the foot showed that the mineral matter had been entirely removed from the phalangeal and metatarsal bones of the little toe, and that the proximal end of the fourth metatarsal bone had been similarly affected.

The position now seemed to be this, that the exact nature of the disease was still doubtful, but that the disorganisation of the foot necessitated amputation. In this doubtful condition it seemed essential that the amputation should be wide of the disease; and this, combined with the experience of the usefulness of the stump left after a "Syme", seemed to leave no choice but that.

The foot was amputated on December 26th, 1912. The amputation seemed to have removed all trace of disease, though some anxiety was felt on account of a softness in the texture of the surface of the tibia, as exposed by the sawing of the bone. The stump, however, healed by first intention; and, at this

date, 15th March, 1913, the stump is free from tenderness and appears healthy. The health of the lad has remained unimpaired.

It should be mentioned that Colonel Shearer, already referred to, sent full details of the history of the case and a description of its present condition towards the end of 1912, to Sir Ronald Ross. Sir Ronald, expressing the strongest possible scepticism as to the likelihood of a case of Madura Foot originating in this country, did not think that the description was that of Madura Foot; later, however, he was inclined to pronounce a less dogmatic negative, and had difficulty in suggesting what the morbid appearances could be caused by.

As I do not profess Bacteriology, and as the exigencies of private practice and the interest of the patient removed the case at certain stages from my care, I am unable to provide slides or drawings of the microscopic and bacteriological work. The attempt to obtain cultures from the second toe at the time of amputation failed; but, in the opinion of Dr. Anderson, Pathologist to the Victoria Infirmary, Glasgow, microscopic slides gave the characteristic appearance of the Streptothrix Maduræ.

L i t e r a t u r e o f M a d u r a F o o t .

The subject of Madura Foot, complicated as it is by supposed identity with Actinomycosis, is a difficult one to make clear. It is still denied by some authorities that the condition is a bacterial one. at all. Some, as has been said, identify it with Actinomycosis, and some authorities discuss the probability of the disease, when producing black roe-like bodies, being a different disease from that which ~~the~~ produces the pale roe-like bodies.

It is evident that the disease had long been vaguely recognised. The number of names applied to it is an index of the ignorance prevailing regarding its pathology and aetiology. The names by which the disease is known are, first, such as are suggested by its local occurrence, second, by its obvious appearance, and third, by its supposed cause. First, the name, Madura Foot, indicates of course its commonest place of occurrence; second, Morbus Tuberculosis Pedis, Godfrey Eyre's Tubercular Disease, Ulcus Grave, Caries, Hypertrophy with Caries, Perforating Ulcer of Foot describe its naked-eye appearances. Third, the names, Fungus Foot, "Work of Worms", indicate the supposed cause; while such a name as "Insect Eggs" may be taken

to indicate both the appearance of the developed disease and a theory as to the cause by which it is induced. It is not surprising that a disease, so localised as Madura Foot is for a length of time, involving no impairment of general health, should have suggested to a community that has much experience of parasitic worms, that the morbid appearance was caused in that way.

It was in 1874 that Dr. Vandyke Carter published his notable investigation. He endeavoured to establish the existence of two or perhaps three varieties, black, pale and perhaps red. He declared his belief in the bacterial origin of the disease, but did not succeed in identifying the fungus. It fell to later workers, such as Bassini, Boyce, Kanthack and Surveyor, to work out the bacterial nature of the disease. Some are of opinion that the black variety is a mere degeneration, and that the blackness is due to pigment, some have contended that the roe-like masses themselves are but an excretion, consisting mainly of fat.

Carter fully discussed the possible causation of the disease. Although many of the Indian natives in whom it is found are underfed, he contended that Anaemia did not predispose to the disease, nor, on the other hand, did plethora. The fact that Indian natives

is disagreeing with some views. Actinomycosis is suffered more from the disease than Europeans he pointed out is not surprising, seeing that they are more employed about the fields, have mud, containing decomposing vegetable and animal matter suspended in it, adhering to their naked feet, and that their feet are more liable to injury than those of Europeans. Many of the natives suffer from foul ulcers, and these, as well as slight abrasions of the foot, might serve to afford entrance to the bacillus; but even this is not necessary. Carter pointed out that, while the diameter of the sweat-gland orifice is about $\frac{1}{2000}$ inch, the size of the Streptothrix is only about $\frac{1}{6000}$ inch, inferring ease of entrance even in a thoroughly healthy condition. The orifice of the sebaceous glands is still larger, and therefore affords even easier access. But the latter glands do not occur on palm or sole, but even the possibility of infection in these areas by the sweat glands still remains. Nay, Carter is of opinion that the bacillus may enter even between the layers of the unbroken epithelium. No doubt, as he points out, the existence of bruised blood immediately beneath the skin will afford a very favourable nest for development. In connection with Stirling's case, it should be specially noted that Carter at one period came to hold the view that Melanoid Mycetoma was identical with Actinomycosis. But there can be little difficulty with

in disagreeing with this view. Actinomycosis is almost unknown in India, Where Madura is more common; Actinomycosis has thoracic and cervical complications; Madura Foot has none; Madura attacks hands and feet especially, Actinomycosis attacks generally the neck, &c., hardly ever the hands and feet, if indeed ever. There are no black, red and pale varieties of Actinomycosis, These more obvious differences were available even in Carter's time. Later authorities, such as Surveyor and Boyce have been able to show that Actinomycosis grows readily in Hydrogen, Madura Foot slowly or not at all; they also differ in the way in which they take up Aniline stain.

The question has been asked whether Madura Foot is a tubercular disease or a specially localised form of Tuberculosis. It can be neither, for in Madura Foot there is no general constitutional invasion, as in Tuberculosis, nor even in the foot are the lesions at all such as are met with in Tuberculosis. The lesions of the bone show no resemblance, nor do those of the skin. The lesions in Madura Foot are certainly "tuberculated" but the lesions in Madura Foot are, especially in the early stage, mainly in the skin, unlike those of Tuberculosis. Indeed, what two diseases could be more sharply contrasted? In Madura Foot a great mass, resulting; no such condition in Tuberculosis; in Madura Foot small sinuses discharging granules

or roe-like masses, or masses like poppy seeds or black grains like gunpowder or bodies like fishroe; in Tuberculosis large sinuses giving off the usual purulent discharge; in spite of local disease in Madura Foot the constitutional condition of patient remains good; in Tuberculosis there is the unfailing breakdown of health and progressive emaciation, in Madura ~~Foot~~ Foot there is no family history of similar affections; in Tuberculosis there too often is; after amputation there is in Madura Foot characteristic tunnelling to be seen, there is no such thing in Tuberculosis; finally there is in Madura Foot no special tendency for the disease to attack articular surfaces as in Tuberculosis.

For some time it was believed that the active cause of the disease was a pink mould known as "Chionyphe Carteri". At this time Carter believed that the black variety was a resting state of the fungus. He made attempts to grow the mould and fungus and obtained a pink mould, the "Chionyphe Carteri". But Lewis and Cunningham pointed out that the pink mould grows as luxuriantly in connection with preparations that have been long steeped in alcohol.

It should be noted that on section of a diseased foot the hard tissues and the soft tissues present much in common, numerous lined cavities generally communicating with each other by sinuous channels, soft

tening and excavations not only of carpal and tarsal but also of long bones, these cavities packed with soft, yellowish, fatty or gelatinous substance mixed with roe-like particles.

In examining the literature of the subject one looks in vain for any consecutive account of the progress of the disease in a limb, analogous to what one may find, for instance, in the description given in text-books of the typical progressive history of a case of Tuberculosis of the Lungs. The reasons for this are probably two in number. First, that most of the cases have been well advanced before they came under the notice of competent scientific observers; second, that the length of time required for the disease to run its full course is so great that it has scarcely fallen to any one man to observe the development of the disease ~~throughout~~ from the beginning up to the point of complete destruction of the deep tissues of the foot or hand.

Probably the best systematic account of the disease is to be met with in the work of Allbutt and Rolleston. I shall now subjoin a brief summary of their account of Madura Foot, in order that it may serve as a basis of the concluding section:- "Was Stirling's a case of Madura Foot?"

From that and the other sources already mentioned, and from those to be mentioned in the subjoined Bibliography, it is learned that the chief seat of the disease is, as its name implies, in Madras. It is found also, however, in Ceylon, Egypt, the Sudan, Madagascar, Morocco and Algeria. In the New World in Cuba and on the Continent of America. In Europe it is said that a case has occurred in Italy. It is naturally commoner in agricultural districts. It is said not to attack Europeans, but this is probably due to the habits of the race in regard to covering the feet. It is oftenest found in males of middle age.

It is noteworthy that the Streptothrix has never been found in soil or in vegetation.

The account of the morbid anatomy is obviously that of an advanced case, and is therefore of less interest to us in our closing discussion. It is stated however, that, on section, the whole foot is softened and the tissues, originally hard and soft, are now ~~more~~ more or less homogeneous, showing a network of canals and cysts, ~~up~~ to the size of a pigeon's egg. These connect with each other, and are the spaces that open on the surface of the foot in fistulous, funnelshaped orifices. They are lined with fibrous tissue and contain the roe-like bodies already alluded to. The site of a bone may be represented by a mere cyst.

As I had no opportunity of devoting attention to the microscopic details, I omit any description of these details.

In dealing with the symptoms of a typical case, I shall number the points, in order that later I may consider point by point how far Stirling's case answered to the description.

(1). There is a decided preference for the right foot.

(2). There appears an indurated circumscribed swelling, about the size of a threepenny piece.

(3). In two months this softens or breaks down, a fistulous opening appears in the centre, giving off purulent, viscid, oily discharge with granules, red, yellow or black.

(4). New nodules appear, making the skin of the foot irregular and tuberculated; ~~the~~ size of nodules from shot up to filbert.

(5). Foot becomes two or three times its proper breadth. The sides become rounded, making the foot appear oval. The arch disappears, the sole becomes convex; the toes are raised, so that the heel and toes cannot touch the ground at once.

(6). Ends of fibula and tibia become thickened, so that the ankle seems swollen.

(7). The skin now becomes elastic and the whole

foot feels homogeneous, due to the harder tissues having become softened.

(8). A probe introduced into a fistula can be passed in all directions without resistance.

(9). Skin may be sensitive, but generally the deeper tissues are insensitive.

(10). Leg above thin and atrophied.

(11). Popliteal and inguinal glands may be enlarged, due, not to metastasis but to septic absorption.

(12). The disease may occur in knee, hands, neck or jaw.

(13). It may run a course of twenty years or more till death from exhaustion or intercurrent affection.

(14). Iodide of Potassium has not been found of service. Early and wide removal of the disease being the only safe course.

Graphs in this case were taken at intervals of six months, and showed a gradual increase in the swelling of the foot. There was no large and deep ulcer as in Madura Foot, leading to sloughed surface, pus, etc.; the open places of this foot healed till near the end to be almost normal, and the ulcers were of the slightest. There was nothing to suggest an Elephantiasis Foot. Iodide of Potassium is said to be useless in Madura Foot; it appeared to delay the spread of the disease in this case.

W a s S t i r l i n g ' s a C a s e
o f M A D U R A F O O T ?

It is certainly much to be regretted that a case should have been so long under observation without a positive diagnosis having been arrived at. But much can be said in extenuation. The disease is looked upon as preeminently one of tropical climates, and ~~as~~ especially as of Indian origin. Stirling had never been out of Scotland. The prevailing opinion was that it was not possible for the Streptothrix to develop in our climate; and Sir Ronald Ross, who was consulted at a late stage of the case, and who expressed himself as most sceptical as to the Madura character of the disease (though later this view was less strongly expressed) was of this opinion. The disease as described and depicted in text-books and monographs is accompanied with much swelling and distortion; till the latest stage of this case there was hardly any swelling or distortion. There are large and deep sinuses in Madura Foot, leading to sloughed tendons, muscles, &c.; the deep tissues of this foot seemed till near the end to be almost normal, and the sinuses were of the slightest. There was nothing to suggest an Elephantiasis foot. Iodide of Potassium is said to be useless in Madura Foot; it appeared to delay the spread of the disease in this case.

These considerations, however, I now see to have been accorded too great weight; the fact is that none of us was able to realise the extreme chronicity of the disease. Although to us the disease seemed to have lasted a great length of time two years was but a short time in the life-history of a case of Madura Foot-. We ought not to have expected to find the characteristic appearances of a fully developed case in one that was only beginning. Further, did not the disease progress more slowly and develop less characteristic manifestations in a European and in a temperate climate?

The surrounding circumstances were peculiar. The trouble on the foot seemed to originate after a kick. This idea was naturally prominent at first, and prevented the bacterial possibilities being taken into account. When the possibility of a tropical disease was mooted it was recalled that Stirling had spent successive summers, as already indicated, in Arran, at a rather ^{dirty} farm, occupied by a man who had spent some years in Australia. This man had brought home with him certain weapons, with which Stirling often played. On many occasions Stirling ran about in Arran barefoot. But the Arran sojourn again introduced another complication. The idea was several times mooted that the case was one of Actinomycosis. During the summer of 1910, during Stirling's holiday in Arran, some

months before the first manifestation of the disease, there was a litter of pups, one of which had a large abscess on the jaw, which burst and discharged pus for quite a length of time. Hence in the less developed condition the possibility of Actinomycosis could not be lost sight of.

The idea of Madura Foot was early suggested along with every other foreign and uncommon malady, and it is curious that competent observers failed to find the Streptothrix, although preparing a self-vaccine. The possibility of Madura Foot was suggested to one after another of the medical persons with tropical experience who saw the foot, but each unhesitatingly declared the appearances unlike any Madura Foot he or she had seen. Yet it was observed throughout the case that no one would venture on an alternative diagnosis. The unfailing response alike of those experienced in Madura Foot and of those who were not so experienced was that they had never seen any affection in the least resembling it.

If it was a case of Madura Foot how was it acquired? Even those familiar with the persistence of life of certain bacilli will have great difficulty in believing that the germs were conveyed from Australia, (where it is hardly known to exist) to Arran, adhering for years to weapons, &c., a most unsuitable

nidus. Could the bacillus have survived the rigours of the intervening winters? That the poison could have been conveyed to this country on bananas is a suggestion that is not likely to be entertained.

The only tentative diagnosis suggested was that of a tubercular or specific affection. But the entire absence of constitutional manifestations of any kind in this prolonged illness negatives such a suggestion. The idea was again raised in connection with the opaque body seen at the side of the spine in the skiagram of that part, and the opinion was expressed that the shaded oval body with a black spot was a tubercular gland with a calcified spot. But even if that were so (and it was not proved to be true) it is an entirely insufficient foundation on which to base such a diagnosis.

To conclude then, as was suggested, by a comparison of the points numbered above:-

(1). The disease usually occurs in the right foot of a male. It was so here.

(2). It began with an indurated, circumscribed swelling. It did so here.

(3). This breaks down, leaving a fistulous opening from which comes a viscid fluid, with coloured granules. The granules were strikingly absent here, but on the side of the foot and on the edge of the

nail of the great toe, there was a breaking down with viscid discharge. At the side of the foot, -but there only- was there a tendency to fistula formation, and that slightly.

(4). New nodules appear. These appeared here, but only at edge of nails of fifth, then of second, third and fourth toes.

(5). Broadening of foot and rounding of sides with disappearance of arch. This was not a prominent feature here; doubtless there was no time for its development.

(6). Ends of tibia and fibula enlarge. There was doubtless a slight enlargement, but it was more apparent than real, owing to atrophy of muscles.

(7). Elasticity of skin and homogeneity of foot had not time to appear.

(8). Painless and easy introduction of probe. There was never a fistula large enough to introduce a probe, and the diseased surface was exquisitely painful.

(9). Sensitive skin. Insensitive deeper structures. Both features were present.

(10). The atrophy of leg was extreme.

(11). The popliteal and inguinal glands affected. These glands entirely escaped; but there seemed to be an enlarged lymphatic gland in front of ankle.

(14). Iodide of Potassium appears to be of no benefit. It is difficult to believe that it had not a favourable effect in this case.

in addition to the submitted two photographs of the leg

C o n c l u s i o n.

That this was a case of Madura Foot in an early stage, there seems no reasonable ground to doubt. The positive evidence adduced is as strong as could be looked for in a case that had not had ~~the~~ time to develop. As for an alternative diagnosis, absolutely none was offered.

It is to be regretted that the condition was not earlier recognised. This would have saved a year of the boy's time, as well as much anxiety and expense. But it can hardly be suggested that earlier recognition would have saved the foot. Even in the earliest stage no one would have taken the responsibility of milder measures than amputation wide of the disease.

I am of opinion that Iodide of Potassium in large doses was serviceable. I cannot say that there was clear evidence of the beneficial effect of the vaccine. No suggestion as to mode of infection can be offered.

P o s t s c r i p t.

Notes of Wagner's Foot recorded in America.

I have stated my intention of supplying, in addition to the subjoined two photographs of the leg and foot, a skiagram of the spine, &c., and a skiagram of the foot, showing the absorption of mineral matter from the affected bones. As the skiagrams were larger than the limits of size laid down for the thesis, I endeavoured to have them reduced; but it proved impracticable to obtain sufficient clearness and definition in this reduced form. The skiagrams are still in my possession.

Cases of Madura Foot recorded in America.

Dr. J. T. Arwine & Dr. Lamb describe a case of Fungous Foot, occurring in America. It was a male, aged 45, twelve years chronicity, left side.

They refer to the previously reported cases subjoined:-

Kirkpatrick, Transactions Assoc. Amer. Phys. 1895, Vol. X. page 92. Male, aged 24, Canada, Ten years chronicity, Right side.

Hyde & Senn. Jour. Cut. & Urin. Dis. 1895, Vol. XIV. Page 1. Male, aged 20, Thirteen years chronicity, Left side.

Pope & Lamb. New York Med. Jour. 1896. Vol. LXIV. p. 386. Male, aged 27. Eight years chronicity. Right side.

Wright. Trans. Assoc. Amer. Phys. 1896. Vol. XIII. p. 471. Female, aged 26. 6 Months Chronicity. Left side.

London, 1893. Extract from the Proceedings of the Royal Society of London 1893, 110-112.

Madura Disease, 8vo, London, 1893. Reprint from Lancet, London, 1894, II 639.

Carver Vandyke, On Mycetozoa, London 1891.

B I B L I O G R A P H Y.

In addition to consulting such works as Green's Encyclopedia & Dictionary of Medicine & Surgery, Osler's Textbook and Allbutt's Textbook, I have consulted the following:-

Adami, J.G., and Kirkpatrick, B.A. Notes upon a case of Madura Foot occurring in Canada. Reprinted Transactions of the Association of American Physicians, 1895.

Bassini. Arch. Pour la Sci. Med. XII No15, p.309.

Bocarro, J.E. Mycetoma. Indian Medical Record, Calcutta, 1893, vol. I, 74, 106, 148, 214.

Further Clinical Notes and Remarks on Mycetoma. Indian Medical Record VI, 1894, 332-334.

An Analysis of One Hundred Cases of Mycetoma. Lancet, London, 1893, II, 797.

Clinical Notes on Mycetoma differentiating the pale and the black variety. Indian Medico-Chirurg. Review, Bombay, 1894, II, 894-896.

Boyce R. W. and Surveyor N.F. Upon the existence of more than one fungus in Madura Disease (Mycetoma)- London, 1893, Cutting from the Proceedings of the Royal Society of London 1893, ~~4111.~~ 110-112.

Madura Disease, 8vo, London, 1893. Reprint from Lancet, London, 1894, II 639.

Carter Vandyke, On Mycetoma, London 1893.



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